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False Localization of Ictal Activity by Scalp EEG in Candidates for Hemispherectomy

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Two patients with intractable seizures and large structural lesions were candidates for hemispherectomy for seizure control. Repeated ictal EEGs recorded from scalp falsely localized seizure onset to the contralateral hemisphere. Intracarotid amobarbital study was performed during left-body epilepsy partialis continua in one subject. Amobarbital was injected in the right internal carotid artery, despite electrographic seizure activity apparently originating in the left hemisphere. Clinical and electrographic seizure activity ceased immediately at the onset of left hemiparesis, without inducing right hemiparesis, speech loss, or EEG attenuation on the left. Intracranial recordings in both patients confirmed that the clinically suspect hemisphere was the source of the seizures, and hemispherectomy produced excellent clinical results. In the presence of a large structural lesion, it appears that scalp EEG may falsely localize ictal discharges to the contralateral side. **Key Words:** Hemispherectomy—False localization—Subdural electrocorticography—Intractable epilepsy—EEG videotelemetry.

Children with medically intractable epilepsy who are candidates for surgical treatment with hemispherectomy undergo extensive preoperative evaluation to confirm their appropriateness for this procedure. This includes a careful clinical history of the seizures, neurological examination, neuroimaging, and interictal and ictal scalp EEG. These studies are supplemented when necessary by either intraoperative or extraoperative electrocorticography (ECoG). Most surgical epilepsy centers require that all available evidence agree on localization of lesions and seizure onset if surgery is to proceed (1). We report two patients with intractable seizures who

were evaluated for hemispherectomy. Both had large structural lesions in one hemisphere. The clinical characteristics of the seizures and the associated neurological deficit both implicated the damaged hemisphere as the source of the seizures. However, repeated ictal EEG recordings pointed to the contralateral hemisphere as the origin of the seizures. This phenomenon of consistently falsely localizing scalp EEG in the presence of large structural lesions has been reported to date only in abstract form (2).

Case Reports

Case 1

At 8 years of age, this patient experienced the acute onset of partial simple seizures, involving left arm, face, and leg, alone and in combination. Left hemiparesis, initially thought to be a Todd's paralysis, was reported after the onset of the seizures. Initial evaluation elsewhere included spinal fluid examination and carotid angiography, both normal. Com-

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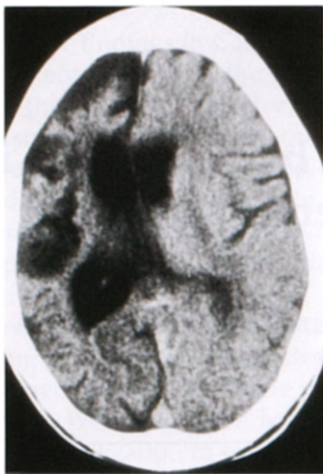


Figure 1. CT scan at the level of the lateral ventricles shows marked cystic encephalomalacia involving the entire right hemisphere.

puted tomography (CT) shortly after the onset of seizures showed edema of the right hemisphere. He was hospitalized for most of the subsequent 2 years for seizures, obtundation due to medication, and behavior problems. At age 10, he had a dense left hemiparesis with secondary scoliosis, left hemi-sensory neglect, and visuospatial deficits. CT performed approximately 2 years after onset of seizures showed severe right hemisphere cystic encephalomalacia (Fig. 1). Cognitive function had reportedly declined from prior to seizure onset, but earlier abilities and achievement were not well-documented. Full-scale IQ was approximately 70.

Seizures were partial simple, with clonic movements of the left arm, often spreading to include the left leg and face, and occasionally interfering with speech, but not comprehension. Seizures were nearly continuous while awake. His *epilepsia partialis continua* (EPC) could only be stopped with massive sedation. On several occasions, barbiturate coma temporarily controlled EPC, which recurred on withdrawal, despite high doses of other anticonvulsants. He was treated with barbiturates, phenytoin, mesantoin, carbamazepine, valproic acid, and various benzodiazepines, alone and in combination. Management was complicated by mesantoin-induced pseudolymphoma syndrome and severe carbamazepine-induced agranulocytosis. Numerous ictal EEG recordings showed irregular sharp waves and spike-wave discharges over the *left* hemisphere, or bifrontal synchronous spikes at seizure onset.

Intracarotid amobarbital injection (Wada test) with continuous scalp EEG was performed. Amobarbital (100 mg) was injected into the right carotid just

after onset of a typical seizure with left arm clonic movement. EEG showed rapid spikes beginning on the left, quickly becoming bilateral (Fig. 2). With right carotid amobarbital infusion, left hemiplegia developed, the seizure stopped, and bilateral scalp EEG ictal activity ceased, without effect on speech, right hand use, or memory.

The patient underwent a classic right hemispherectomy with intraoperative ECoG. Continuous ictal discharges were documented diffusely from the right hemisphere. When examined pathologically, the cortex showed widespread cystic changes with widespread gliosis, necrosis, and perivascular cuffing, consistent with Rasmussen's encephalitis.

Postoperatively, the patient had a single seizure. He had no change in his left hemiparesis but had a new left homonymous hemianopia. He had a gradual improvement in behavior and was able to return to public school for the first time in over 3 years. He was tapered off anticonvulsants 3 years after surgery without seizure recurrence.

Case 2

This patient, now 24 years old, had had seizures from 2 years of age. She was the product of a normal pregnancy, labor, and delivery. She had no notable illnesses. Development was delayed. She walked at 2 years, spoke in single words at 3 years, and has always required special education. She functioned in the moderately retarded range, with independent self-care and ambulation when well. Hydrocephalus due to atypical Dandy-Walker malformation was diagnosed and treated with a ventriculoperitoneal shunt at age 11, with several subsequent shunt revisions. She had life-long severe right hemiparesis, right hemiatrophy, and a right homonymous hemianopia, with moderate left-sided hyperreflexia. She was evaluated for hemispherectomy at age 21 years because frequent seizures were impairing her abilities to walk and perform self-care tasks. She had several seizure types: simple partial seizures, with right arm extension and head to right, complex partial with undressing or other automatisms, and secondarily generalized seizures, beginning with right-arm tonic activity. Frequency over 8 years of vigorous treatment varied from 2–3 per week to 8–10 per day. She received various combinations of phenytoin, phenobarbital, carbamazepine, valproic acid, primidone, and acetazolamide. Magnetic resonance imaging demonstrated marked thinning of the left hemisphere, hydrocephalus, a retrocerebellar cyst, and hypoplasia of the vermis (Fig. 3). The skull was markedly thickened.

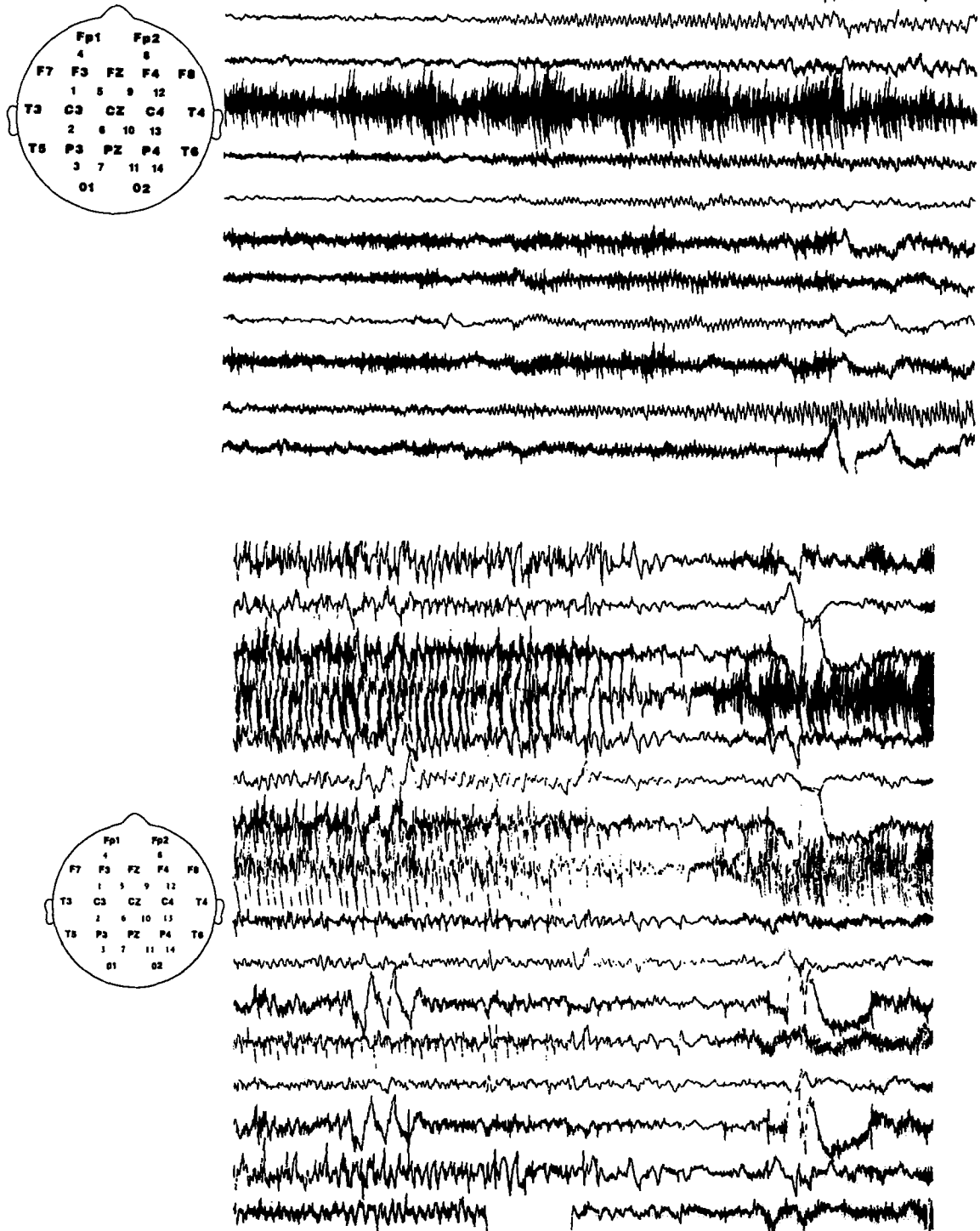


Figure 2. *Top:* Case 1. EEG recorded during Wada test: first arrow indicates onset of left arm clonic jerking, followed by injection of amobarbital in right carotid. *Bottom:* EEG recorded 60 s after amobarbital injection. Clinical and electrographic seizure activity stops and left hemiplegia is observed, without loss of speech or right hemiparesis.



Figure 3. Case 2. MRI (spin echo; TE, 465/TR, 26) demonstrates ventricular enlargement and marked thinning of the cortex of the left hemisphere.

Her interictal scalp EEG showed near-continuous spikes and high-amplitude slow waves emanating from the left hemisphere. However, onset of seizure discharge during videotelemetry was always in the right hemisphere (Fig. 4).

Intracarotid amobarbital injections were performed. Amobarbital injection (100 mg) into the right internal carotid artery produced a left hemiplegia and speech arrest, with no change in interictal epileptiform activity. Left internal carotid injection (125 mg) did not change speech or already severe right arm paresis. EEG showed left-sided voltage attenuation and suppression of interictal epileptiform discharges.

Subdural strip electrodes were placed to map seizure origin. Strip electrodes were placed through burr holes over the lateral frontal, lateral temporal, subtemporal, and subfrontal lobe areas bilaterally. Interictally, there were continuous epileptiform discharges from the left frontal and temporal cortex. The right hemisphere showed no electrographic epileptiform activity. Ictal onset was shown to be consistently from the left lateral frontal, lateral temporal, and subfrontal leads. The right hemisphere was quiet during seizures recorded intracranially (Fig. 5).

A modified left hemispherectomy was performed. Pathological examination demonstrated a dysplastic hemisphere with multiple heterotopias. The hemisphere was extremely thin, with abnormalities of cortical lamination and very deficient white matter.

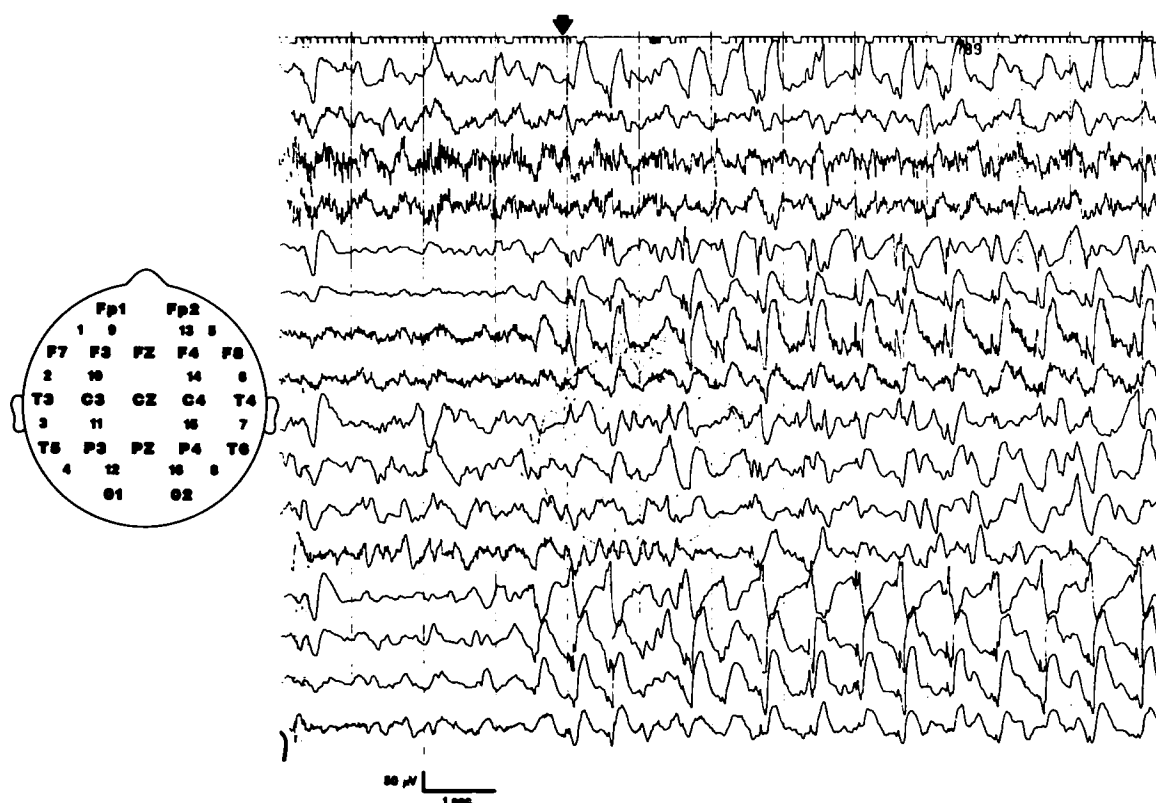


Figure 4. Case 2. EEG during videotelemetry: epileptiform discharge begins in right hemisphere, at onset of clinical seizure activity involving right arm (arrow).

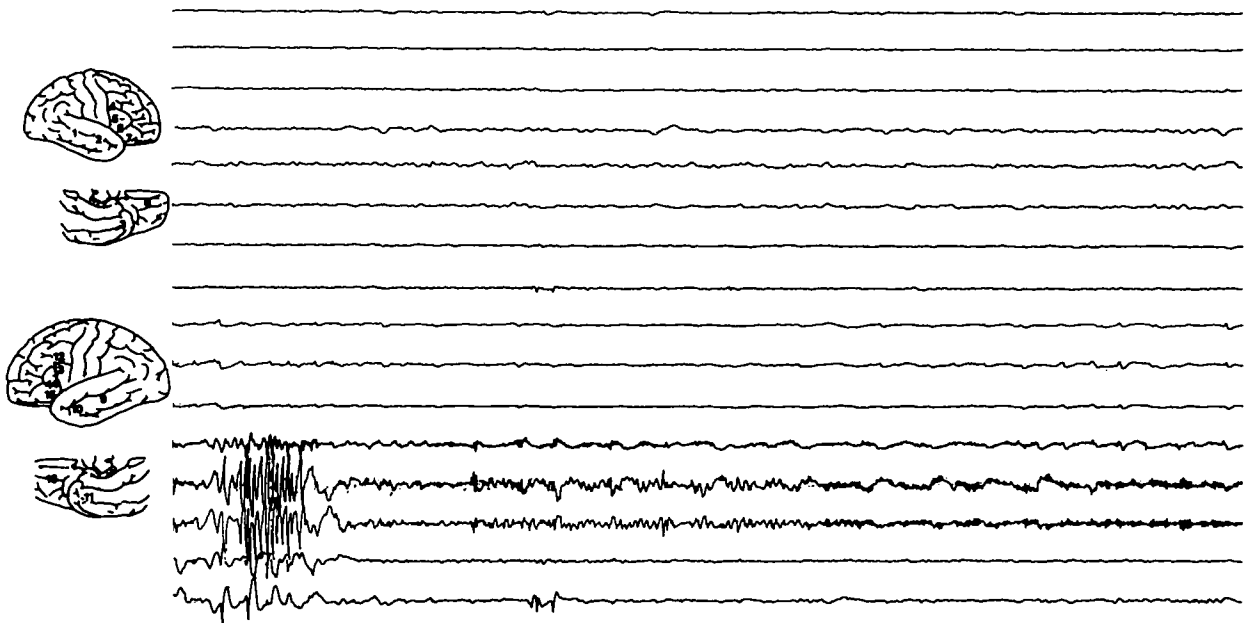


Figure 5. Case 2. Seizure recorded using subdural strips documents electrographic seizure onset from left hemisphere, without right hemisphere involvement.

Seizures were markedly reduced 18 months postoperatively and consist only of head and eye deviation lasting 2–3 s. There was no change in her right hemiparesis and hemianopia. Her gait improved, and she became independent in activities of daily living, and began attending a sheltered workshop.

Discussion

False localization of ictal onset from scalp EEG has recently been discussed in detail in candidates for temporal lobectomy (3). Scalp EEG may either demonstrate bitemporal interictal abnormalities in the face of unilateral ictal onset on depth EEG or may fail to demonstrate bitemporal abnormalities when present. Wyler et al. (4) compared ictal scalp EEG to subdural strip recordings in 120 subjects who had good results from surgical treatment. They found consistent false lateralization of ictal activity by scalp recording in 5 of 120 subjects, 2 of 108 with temporal resections, and 3 of 16 with extratemporal cortical resections. Engel and Crandall (5) reported falsely localizing ictal onset from depth EEG when anticonvulsants were withdrawn during prolonged monitoring. However, ictal onset was seen both at the lesion site, producing "typical" seizures, and elsewhere, producing "atypical" episodes. Lee et al. (6) recently reported a patient with consistent false localization of ictal onset by scalp EEG in a patient with temporal lobe seizures without a radiographically apparent structural lesion. It is notable that in both of

our patients, scalp EEG consistently localized ictal onset *contralateral* to large and clinically obvious lesions. This occurred both on full and reduced dosage of antiepileptic medication. On scalp EEG, ictal onset was never recorded from the hemisphere that proved to harbor the actual focus, despite repeated monitoring.

We conclude that ictal scalp EEG recordings may be misleading. Despite numerous recordings, scalp EEG falsely localized seizure onset in both patients. The reason for false localization of the scalp EEG is not clear, particularly since correct localization was easily established on intraoperative electrocorticography or subdural recording.

Although the mechanism of this false localization is uncertain, one possibility is that it may be due to the distribution of voltages in a volume conductor. The ultimate voltages and their distribution as recorded from scalp EEG depend on the summation of a number of primary voltages with differing strength, position and orientation, and the electrical and geometrical properties of the substance in which the sources are embedded (7–9). It is the latter that is probably responsible for the phenomenon of false localization in scalp recordings. Cerebrospinal fluid (CSF) has a very low resistance compared to surrounding brain and dura. Therefore, CSF overlying the brain is likely to act as a region of high conductivity shunting the current before it has an opportunity to traverse the adjacent high-resistance region composed of dura, bone, and skin (10). In the pres-

ence of a seizure focus embedded in a markedly atrophic hemisphere where there is excess CSF over the focus, the excess CSF could conduct the discharges to the contralateral side. This phenomenon, of course, would be eliminated during subdural recordings, and accurate localization would be assured.

Our experience suggests that further evaluation and surgery are indicated when ictal scalp EEG localizes to the hemisphere contralateral to large lesions. We agree with Wyler et al.'s conclusion that "decisions based on scalp monitoring will result in excluding numerous patients who are excellent surgical candidates" (4). Had apparent ictal onset contralateral to the lesion been considered a surgical contraindication, both of these patients would have been denied surgical therapy that proved to be extremely effective.

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